Environmental Health Perspectives
Vol. 30, pp. 171–172, 1979

Experimental Approach to the Determination of Pulmonary Carcinogenic Influences of Shale Oil Effluents

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Oil derived from oil shale deposits is known to contain many organic complexes. The formation of carcinogenic hydrocarbons is temperature-dependent and is associated with retorting of the oil. Furthermore, oil shale is a rich source of inorganic elements such as the metals. Biological studies have demonstrated that concentrated extract of tars from combustion of shale oil are carcinogenic to the skin of mice. The purpose of the current project is to evaluate the potential carcinogenic hazard from inhalation of retort and combustion effluents for man. These studies will be carried out in pathogen-free rats by intratracheal instillation with and without added factors such as supplemental particles and known carcinogens as interactants.

Present and predicted shortfalls in the production of petroleum necessitate development of substitute sources for liquid fuel on an unprecedented level. The commercial extraction of liquid hydrocarbon from oil shale is a technology that is being carried out in the Soviet Union, Brazil, and other areas. Since enormous reserves of this mineral exist in the United States, preliminary feasibility studies for its exploration are being carried on here.

Extraction and refining of keragens from oil shale is a complex procedure in which mining, retorting, and refining into various products by distillation result in a number of potential threats to human health. These threats consist of airborne dust from mining and crushing operations, vapors emitted from retorting and refining, and emissions from the combustion of the refined product for the production of power. These potential hazards must be evaluated from the standpoint of possible injury to the health of industrial workers, miners, processors, and engine operators, as well as risk to the community population both as point source air pollutants and as contributors to the general atmospheric burden of potentially toxic chemicals.

Oil shale is a rich source of trace metals and contains very complex organic matter. Furthermore, the formation of carcinogenic hydrocarbon is known to take place during the extraction of the crude from the rock during retorting and is known to be temperature-dependent (*1*). Little is known at this stage concerning the potential of refined American shale oil products to emit hydrocarbon carcinogens during combustion in stationary power stations or various forms of engines such as internal combustion, turbines, or jets. Other factors to consider are the presence of interacting carcinogens or promoters such as the metals, phenols, etc. (*2, 3*).

The study of the carcinogenicity of combustion products in general has been carried out on particulate emissions collected directly from sources such as coal burning, automobile emissions, and the ambient air. These studies have usually indicated that the crude organic extracts and a number of component fractions or subfractions are capable of eliciting cancers from percutaneous application to the skin, cutaneous injections, and inoculation of newborn mice (*4, 5*).

While the concentrated extracts of tars from such combustion sources have been capable of eliciting

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tumors of the skin or in neonate mice, such material has been generally unsuccessful in the production of pulmonary tumors (*6*). However, early work found crude particulate material, such as chimney soot and road dust, capable of eliciting experimental tumors of the lung (*7, 8*). Subsequently, experiments in which pure carcinogens were mixed with various particles were regularly successful in the production of pulmonary tumors (*9, 10*). It would thus appear that either crude carcinogen-containing material or refined products with added particles are requisite for experimental production of pulmonary tumors with polycyclic hydrocarbons.

It is now known that furnaces burning coal and oil emit large amounts of polycyclic organic matter containing carcinogenic activity (*11*). Emissions from vehicles fired by gasoline or diesel oil contain similar material (*11*). It is highly likely that combustion of shale oil will have somewhat similar tendencies. Several studies with airborne particles have indicated that the whole organic complex may yield more activity than the sum of its component fractions, or that could be expected on the basis of its benzo(a)pyrene content (*4, 5*). This also has been noted for the carcinogenicity of automobile exhaust emissions (*12*). Metals have been considered either as prime agents or interactant inducing agents on the basis of epidemiological observations (*13*), so therefore it would seem appropriate to include metals in carcinogenesis studies involving combustion emissions.

It seems clear that combustion products of carboniferous fuels contribute to the rate of lung cancer (*6*). It is known that the various fuels differ in their contribution of polycyclic organic material and presumably their cancer-inducing potential. It is therefore germane to examine the contribution of shale oil combustion as a part of this total process.

A study is under way in the Environmental Protection Agency's program that is evaluating the potential carcinogenicity of asbestiform minerals released through mining and benefication of metallic ores. These studies concern the carcinogenic potential of fibrous silicate rock and its synergistic reaction with various organic materials, as compared to standard reference amosite asbestos. In these studies in an experimental model based on the enhancement of the carcinogenic activity by the interaction between organic carcinogens and particulates (*9, 10*), the mineral fibers are being considered either as directly carcinogenic or as cofactors in conjunction with organic materials.

Retort effluents and combustion products of shale oil will be studied in conjunction with the above mentioned program in a similar model, in which the effluent will be considered as a prime carcinogen or as a cocarcinogen as previously reported for oil shale soot in Estonia (*14*). The approach will be as follows. Effluent from retorting of oil shale rock and combustion products of various refined shale oil products will be collected on filters, prepared for intratracheal instillation in gelsaline and injected into the lungs of pathogen-free rats. Identical samples will be subject to chemical analysis by others in the shale oil program. Additional experiments will be done employing shale oil effluents in conjunction with benzo(a)pyrene and other organic carcinogens. In this way, we hope to determine whether the effluents have carcinogenic and/or cocarcinogenic potential.

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